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DYSBARISM. HIGH-ALTITUDE ILLNESS

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Dysbarism is the collective term used to describe the pathologic changes that occur when the human body is exposed to environmental pressure changes (alternobaric exposure). Those altered pressures are translated into unphysiologic behavior of gases in organs and tissues. Failure to adequately or timely adapt to those changes, can generate (depending on a number of exposure and individual factors) the different clinical syndromes of dysbarism. Alternobaric exposure is a concern in a number of occupational and recreational activities, such as diving, compressed air work (as in tunnel construction and caisson work), as well as in aviation, mountain climbing, and high-altitude flying.

I. Barotrauma. Barotrauma is the most common medical problem of divers. It refers to bodily injury resulting from changes in ambient pressure. Barotrauma commonly involves the ears, sinuses, and lungs, although any closed air space may be involved.

A. Barotrauma of descent. Barotrauma of descent occurs when trapped air is compressed on descent, resulting in contraction and deformation of the surrounding tissues.

1. External ear squeeze (barotitis externa) occurs when air is trapped in the auditory canal, usually by cerumen, ear plugs, or a tight-fitting hood. The canal is deformed inward and the tympanic membrane is drawn outward, sometimes to the point of rupture. Symptoms include pain and bloody discharge. Otoscopic findings may include canal edema and hemorrhage and tympanic membrane perforation. Treatment includes analgesia, keeping the ear dry, and administration of an oral antibiotic and otolaryngologic referral if perforation is present.

2. Middle ear squeeze (barotitis media) occurs when air trapped in the middle ear is reduced in volume by the increased pressure of descent. Hemorrhage, edema, and inward rupture of the tympanic membrane may occur if equilibration through the eustachian tube does not occur. Treatment is similar to that of external ear squeeze (see sec. I.A.1 above), with the addition of systemic and nasal decongestants.

3. Internal ear squeeze (barotitis interna) occurs when a pressure differential results in rupture of the oval or round window. Patients experience tinnitus, vertigo, and a sensorineural or mixed hearing loss. Management includes hospitalization, bed rest with the head elevated, and otolaryngologic consultation regarding possible repair of a perilymph fistula.

4. Sinus squeeze most often involves the frontal and maxillary sinuses and occurs when the aerated space volumes are decreased due to increased pressure. Mucosal hemorrhage and edema, pain, and epistaxis are common. Treatment includes analgesia and administration of systemic and nasal decongestants.

5. Suit squeeze and mask squeeze occur when tissue is deformed by the diminished volume of air trapped in a tight-fitting dry suit or face mask. Findings may include skin erythema, petechiae, ecchymoses, and conjunctival or scleral hemorrhages. Usually no treatment is required.

B. Barotrauma of ascent. Barotrauma of ascent occurs as a result of gas expansion due to decreased ambient pressure as a diver ascends toward the surface.

1. Reverse ear squeeze occurs when air trapped in the middle ear expands, resulting in pain and possibly outward rupture of the tympanic membrane. Treatment is similar to that of middle ear squeeze.

2. Alternobaric vertigo is thought to be due to a large pressure differential between the middle and inner ear or between the two middle ears. It usually occurs on ascent but may occur on descent after a forced Valsalva maneuver. It is characterized by a fullness in the ear followed by transient but often severe vertigo and disorientation. Rarely, a transient facial palsy occurs. Treatment includes analgesia, administration of nasal and systemic decongestants, and sometimes myringotomy to accomplish pressure equilibration.
3. Tooth squeeze (barodontalgia) occurs when air trapped within a tooth expands, resulting in pain and possibly damage to fillings. Treatment includes analgesia (including anesthetic infiltration) and in severe cases chamber recompression.
4. Pulmonary barotrauma (pulmonary overpressurization syndrome [POPS]) can occur from ascent from depths exceeding 1 m if exhalation does not take place during ascent or if there is pulmonary air trapping secondary to lung disease. Under these conditions, as a result of decreasing ambient pressures encountered during ascent, overdistention of the lungs occurs. At transpulmonic pressure gradients in excess of 80 mm Hg, rupture of the lung parenchyma and pulmonary vasculature may occur. This can result in four syndromes, singly or in combination, that usually are present upon surfacing. Each generally requires hospitalization.
a. Pulmonary tissue damage is manifested by cough, dyspnea, and hemoptysis. Treatment includes supportive measures, particularly administration of oxygen by mask. Positive pressure ventilation generally should be avoided because of the risk of causing further damage to the lungs.
b. Soft-tissue emphysema may involve the subcutaneous tissues, mediastinum, and pericardium. Physical examination and radiologic evaluation will establish the diagnosis. Treatment is supportive care, including administration of oxygen by mask.
c. Pneumothorax is manifested by cough, dyspnea, and pain. The diagnosis is confirmed by a chest radiograph. Treatment generally involves tube thoracostomy, although small pneumothoraces may be managed by careful observation.
d. Air embolism occurs when pulmonary air enters injured pulmonary vessels and gains access to the systemic circulation through the left heart. Air emboli block distal circulation, producing ischemia and infarction of the involved tissue. Any organ may be involved, but the most serious syndromes are as follows:
(1) Coronary air embolism produces typical signs and symptoms of myocardial ischemia and infarction. Management involves treating the myocardial ischemia or infarction and air embolism.
(2) Cerebral air embolism
(a) Manifestations appear abruptly upon surfacing and include headache, dizziness, visual disturbances, sensory disturbances, monoplegia, hemiplegia, an altered mental status (e.g., personality change, confusion, coma), seizures, and death.
(b) Treatment
(I) The patient is placed supine or, if possible, in the Trendelenburg and left lateral decubitus positions to lessen the risk of additional cerebral emboli and improve the cerebral circulation.
(II) Administration of 100% oxygen by mask or endotracheal tube will enhance oxygenation of tissues and increase the gradient for nitrogen elimination from the emboli.
(III) Intravenous fluids are administered as needed to achieve adequate hydration.
(IV) Recompression in a hyperbaric chamber is definitive therapy. If a hyperbaric chamber is unavailable, the patient must be transferred to a facility having a chamber. If air transport is required, it must be in an aircraft that is maximally pressurized, and/or the aircraft must maintain the lowest possible flying altitude.
II. Decompression sickness
Decompression sickness (the bends, caisson disease) is caused by formation of nitrogen bubbles in the blood and tissue following ascent from a dive.
A. Pathophysiology
At increased ambient pressures, the inert nitrogen gas from the compressed air that the diver breathes is dissolved in various body tissues with a predilection for tissues high in

lipid content. The amount of nitrogen dissolved during a dive is a function of both the ambient pressure and the time at that pressure, that is, the depth and duration of the dive. The U.S. Navy Standard Decompression Tables specify safety limits regarding times at various depths and the rate of ascent (i.e., the number and duration of decompression stops) from such depths. Generally, decompression sickness does not occur with dives to depths less than 10 m (33 ft) but may occur with deeper dives when the decompression tables are not followed and, uncommonly, even when they are followed because of individual variability. In such situations, various body tissues are saturated with nitrogen; with too rapid a decline in ambient pressure upon ascent, nitrogen bubbles form in those tissues and in the blood, giving rise to the manifestations of decompression sickness (as a result of the mechanical effects of the bubbles themselves or tissue ischemia).

B. Clinical features

The manifestations of decompression sickness generally appear during the first hour after surfacing but may occur during ascent or be delayed for up to 36 hours. Decompression sickness is usually divided into two types based on the clinical features.

1. Type I decompression sickness refers to skin and/or musculoskeletal involvement only, manifested as pruritus with or without a rash (usually mottling) and pain, respectively.

2. Type II decompression sickness refers to involvement of critical organs, particularly the CNS and lungs.

a. CNS manifestations arise from involvement of the brain and spinal cord. Involvement of the brain may give rise to nausea and vomiting; visual disturbances; tinnitus, vertigo, and nystagmus (the "staggers"); dysarthria; an altered mental status; seizures; and hemiparesis. Spinal cord involvement may yield extremity weakness or numbness, paresthesias, paraparesis, ataxia, and bladder dysfunction.

b. Pulmonary involvement (the "chokes") is manifested by chest pain, cough, and dyspnea.

c. In very severe cases, shock (presumably due to obstruction of the pulmonary circulation by nitrogen bubbles or hypovolemia secondary to increased vascular permeability) and even death may ensue.

C. Treatment

1. In type II decompression sickness, the patient should be placed supine or, if possible, on the left side with the head 30 degrees lower than the feet to decrease the risk of cerebral air embolism and improve the cerebral circulation.

2. To improve tissue oxygenation and enhance nitrogen elimination from the body, 100% oxygen by face mask or endotracheal tube is administered.

3. Recompression in a hyperbaric chamber is essential and should be performed as soon as possible in all suspected cases of decompression sickness with the exception of isolated, mild skin manifestations. If air transport to a facility having a hyperbaric chamber is necessary, the aircraft should be maximally pressurized and/or fly at the lowest possible altitude.

4. Ringer's lactate solution or normal saline is administered IV to achieve and maintain an adequate circulatory volume.

5. Diazepam is the drug of choice for terminating seizures.

6. The role of corticosteroids is controversial, although one retrospective review suggests a beneficial effect.

7. Narcotic analgesics and sedatives should be avoided, if possible, because of their respiratory depressant effects.

D. Disposition

In general, hospitalization for at least 24 hours is indicated for persons with decompression sickness.

III. Nitrogen narcosis

Nitrogen narcosis (rapture of the deep) is a syndrome of delirium, impaired judgment, and diminished psychomotor skills that may occur when compressed air (which is 81% nitrogen) is breathed at a depth of 30 m (99 ft) or more. It results from the anesthetic-like properties of compressed nitrogen and does not occur when helium mixtures are used

instead. It resolves completely with ascent and should not be confused with the behavioral changes seen with decompression sickness or cerebral air embolism.

High-Altitude Illness

Acute exposure to altitudes in excess of 2000 m (6560 ft) may result in high-altitude illness. This illness encompasses a spectrum of clinical entities that likely represent different manifestations of the same disease process. The clinical syndromes of high-altitude illness may overlap and range from mild to severe. Acute mountain sickness, peripheral edema, and retinal hemorrhages generally are self-limited forms of the illness, whereas pulmonary edema and cerebral edema are potentially fatal disorders. Acute mountain sickness, however, may progress to the life-threatening syndromes. Both the incidence and severity of high-altitude illness appear to be directly related to the altitude attained, rate of ascent, amount of physical exertion expended at the high altitude, and individual susceptibility. Thus, high-altitude illness commonly afflicts people who rapidly ascend or reascend (following loss of acclimatization) to a high elevation, particularly if they undergo physical exertion at the high altitude. Manifestations of acute altitude illness generally appear from 6-96 hours after arrival at the high altitude.

I. Acclimatization

In acclimatization, which may be achieved by spending 2-4 days at intermediate altitudes en route to a high altitude, physiologic adaptive processes occur that protect against high-altitude illness. These physiologic processes are complex and principally involve mechanisms that enhance oxygen delivery to tissues in response to the reduced partial pressure of oxygen at altitude. These include the following: (1) an increase in ventilation (resulting in a respiratory alkalosis), (2) renal excretion of bicarbonate to compensate for the respiratory alkalosis, (3) maintenance of cardiac function, (4) pulmonary arterial vasoconstriction to improve matching of ventilation and perfusion, (5) cerebral vasodilatation to enhance oxygen delivery to the brain, (6) an increase in the oxygen-carrying capacity of the blood initially from a hemoconcentration due to a diuresis and later from erythropoiesis, and (7) complex cellular changes to optimize oxygen uptake at the cellular level.

II. Pathophysiologic mechanisms

The pathophysiology of high-altitude illness is complex and not well understood. It appears, however, that the inciting factor is excessive hypoxia due to failure to compensate adequately for the hypobaric-related hypoxia as a result, in part, of a low or blunted hypoxic ventilatory response (and consequent relative hypoventilation) in susceptible individuals. This excessive hypoxia likely leads to abnormal or exaggerated physiologic responses that culminate in the various syndromes of high-altitude illness. Various pathophysiologic mechanisms that may be operative include the following: (1) failure of the adenosine triphosphate-dependent sodium pump leading to intracellular accumulation of sodium and cellular edema (cytotoxic edema), (2) cerebral vasodilatation leading to increased cerebral blood flow (hyperperfusion) and subsequent breakdown of the blood-brain barrier (vasogenic edema), (3) marked pulmonary arterial vasoconstriction resulting in circulatory shear forces and a consequent permeability leak, and (4) antidiuresis possibly mediated by increased antidiuretic hormone and contributing to fluid retention and the various edematous states. It is unclear whether the symptoms of acute mountain sickness are due to increased intracranial pressure secondary to increased cerebral blood volume or represent a mild form of cerebral edema. High-altitude pulmonary edema is a noncardiogenic form of pulmonary edema stemming from a large-pore leak in the alveolar capillary membrane. Retinal hemorrhages may be due to a hypoxia-induced increase in blood flow, with the possible added direct effect of hypoxia on the retinal vessels.

III. Clinical features

High-altitude illness likely represents a single disease with a variety of manifestations that can be organized into five syndromes as follows:

A. Acute mountain sickness

Acute mountain sickness is a symptom complex that occurs with increased frequency at increasing altitudes above 2000 m following rapid ascent. Physical exertion and use of

alcohol or sedatives at high altitude appear to be contributing factors. Signs and symptoms, which usually begin 6-24 hours after arrival at the high altitude, include headache, malaise, anorexia, nausea, vomiting, dizziness, fatigue, weakness, lassitude, poor concentration, insomnia, mild dyspnea, hyperpnea, tachycardia, irritability, periodic (Cheyne-Stokes) respirations, and oliguria. For individuals remaining at the high altitude, symptoms generally resolve in 1-4 days as acclimatization occurs.

B. High-altitude pulmonary edema

High-altitude pulmonary edema, which is a form of noncardiogenic pulmonary edema, generally occurs 1-4 days after rapid ascent to altitudes in excess of 2500 m. There appears to be a predilection for young people and previously acclimatized people reascending to a high altitude following a stay at low altitude. Cold temperatures and physical exertion at high altitude are predisposing factors. Pulmonary signs and symptoms (e.g., cough, dyspnea progressing to severe respiratory distress, tachypnea, frothy pink sputum, rales, cyanosis) predominate, but manifestations of acute mountain sickness may also be present. A low-grade fever, tachycardia, respiratory alkalosis, and leukocytosis are other common features. The chest x-ray reveals patchy infiltrates with a predilection for the right midlung field and a normal-sized heart. The ECG may reveal a right-heart strain pattern reflecting acute pulmonary hypertension. With progression of the disease, an altered mental status, hypotension, and death may ensue.

C. Cerebral edema

Cerebral edema generally requires altitudes in excess of 3500 m for it to occur. Its onset generally is 2-3 days after arrival at such altitudes. Again, rapid ascent is a predisposing factor. Neurologic signs and symptoms are prominent and include a severe headache, ataxia, altered mental status, papilledema, and focal neurologic deficits. Nausea and vomiting are common. With progression of the disease, seizures, depression of consciousness, and death may ensue. Permanent neurologic deficits may be present in survivors.

D. Peripheral edema

Peripheral edema may occur 2-4 days after rapid ascent to altitudes above 2500 m. It is more common in women. The edema generally involves the face initially, progresses to involve the hands and lower extremities, and is associated with a weight gain. Manifestations of acute mountain sickness are often present. The edema usually resolves spontaneously over several days.

E. Retinal hemorrhages

Retinal hemorrhages may occur at altitudes exceeding 4000 m, increasing in frequency the higher the elevation. Physical exertion at high altitude appears to be a predisposing factor. The hemorrhages are usually asymptomatic, but visual blurring and scotoma may be noted with involvement of the macula. Associated features of acute mountain sickness may be present. The hemorrhages usually resolve spontaneously in 7-14 days.

IV. Differential diagnosis

Differential diagnosis includes those disorders that mimic the various high-altitude syndromes. These primarily include dehydration, exhaustion, viral syndrome, gastroenteritis, tension or migraine headache, respiratory infections, meningitis, hypothermia, carbon monoxide poisoning (from using portable stoves or heaters), and thromboembolic events.

V. Treatment

Treatment of high-altitude illness depends on the nature and severity of the disease.

A. Acute mountain sickness

1. Mild acute mountain sickness can be treated by rest at the same altitude to allow for acclimatization and administration of an analgesic (e.g., acetaminophen, aspirin) and an antiemetic (e.g., prochlorperazine) as necessary.

2. For moderate symptoms, acetazolamide, 250 mg PO q8-12h, or dexamethasone, 8 mg initially, followed by 4 mg PO, IM, or IV q6h, can be given. These agents have been shown to be effective in treating acute mountain sickness. Acetazolamide, a carbonic anhydrous inhibitor, by causing renal excretion of bicarbonate and a consequent metabolic acidosis,

stimulates ventilation and improves arterial oxygenation; other beneficial effects in acute mountain sickness include producing a mild diuresis, decreasing production of cerebrospinal fluid, and eliminating periodic breathing during sleep. Possible mechanisms of action of dexamethasone include membrane stabilization, an anti-inflammatory effect, reduction of vasogenic edema, cerebral vasoconstriction, and antiemetic and mood elevation effects.

3. For severe or progressive symptoms, administration of oxygen (if available), drug therapy with acetazolamide and/or dexamethasone, and immediate descent to a lower altitude are indicated. Pressurization in a hyperbaric chamber, if available, simulates descent and can effect resolution of symptoms.

B. High-altitude pulmonary edema

Treatment of high-altitude pulmonary edema includes rest, administration of oxygen, and descent to a lower altitude. If descent is not possible, hyperbaric therapy, if available, can be beneficial. Nifedipine, by reducing pulmonary arterial pressure, may be effective in treating high-altitude pulmonary edema in an initial dose of 10-20 mg SL or PO, followed by administration of a 20-mg sustained-release preparation q6h as needed. Application of continuous or expiratory positive airway pressure (CPAP, EPAP) via a tight-fitting mask has been shown to improve ventilation-perfusion match and oxygenation in this disorder.

C. Cerebral edema

Cerebral edema requires administration of oxygen and immediate and rapid descent to a lower altitude. If descent is not possible, hyperbaric therapy, if available, can be beneficial. Dexamethasone, in an initial dose of 8 mg, followed by 4 mg q6h, is indicated to effect a reduction in cerebral edema. In severe cases, use of a hyperosmolar agent (e.g., mannitol, glycerol) or loop diuretic (e.g., furosemide) may be of benefit. For comatose patients, endotracheal intubation and controlled hyperventilation (to yield a PCO_2 of 25-30 mm Hg at sea level or a lower partial pressure at altitude) are indicated.

D. Peripheral edema

Peripheral edema generally can be treated by rest at the high altitude. Severe or progressive edema, however, should prompt descent to a lower altitude. A diuretic in a low dosage can also be administered.

E. Retinal hemorrhages

Retinal hemorrhages Asymptomatic retinal hemorrhages require no treatment other than rest for a few days at the high altitude. Symptomatic retinal hemorrhages should prompt administration of oxygen, descent to a lower altitude, and ophthalmologic referral.

VI. Prophylaxis

A gradual, staged ascent to high altitude to allow for acclimatization generally will prevent high-altitude illness. Other preventive measures include avoiding overexertion, avoiding respiratory depressants (e.g., alcohol, sedatives), and eating a high carbohydrate diet. Both acetazolamide, 250 mg PO q8-12h or 500 mg of a sustained-release preparation qd, and dezamethasone, 4 mg PO q6h, have been shown to be effective agents for prophylaxis when started 24 hours before ascent and continuing for 48-72 hours at altitude. Although dexamethasone may be the superior prophylactic agent, acetazolamide, which appears to hasten acclimatization, is considered to be the drug of choice because of its lower potential for causing significant side effects. Currently, drug prophylaxis is indicated for persons who have been identified (from past episodes) as being susceptible to developing high-altitude illness or who must ascend rapidly to a high altitude.

VII. Disposition

Patients with mild to moderate acute mountain sickness who improve with rest and treatment may continue their journey to higher altitudes when asymptomatic. Patients with severe or progressive acute mountain sickness, high-altitude pulmonary edema, or high-altitude cerebral edema should descend to a lower altitude and be hospitalized if their symptoms persist.

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Профессиональная патология – клиническая дисциплина, изучающая вопросы этиологии, патогенеза, клинической картины, диагностики, лечения и профилактики профессиональных болезней.

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Гиподинамия;
Перегрузки органов кровообращения, дыхания, голосовых связок;
Нервно-психическая нагрузка

Физические факторы:
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